Clinical Investigation

Serum Potassium Concentration in Hyperglycemia of Diabetes Mellitus With Long-term Dialysis

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Severe hyperkalemia (serum potassium level >6 mmol per liter [mEq per liter]), often with electrocardiographic disturbances, was noted at presentation in 30% of 73 hyperglycemic episodes (serum glucose concentration >25 mmol per liter [455 mg per dl]) observed in 15 in-hospital patients with insulin-dependent diabetes mellitus who were receiving long-term hemodialysis or peritoneal dialysis. Serum glucose concentration and total carbon dioxide content correlated significantly with the presenting serum potassium concentration. Treatment with parenteral insulin alone resulted in a decrease of the serum glucose value from 41 ± 14 (standard deviation) to 11 ± 5 mmol per liter (P <.001) and of serum potassium level from 5.2 ± 1.2 to 4.0 ± 0.6 mmol per liter (P <.001). The changes in serum glucose concentration and in carbon dioxide content and the serum potassium concentration at hyperglycemia were found to be independent correlates of the decrease in potassium concentration during treatment. Insulin alone resulted in correction of hyperkalemia in all instances. Posttreatment hypokalemia was noted in only two instances, each associated with both ketoacidosis and low-normal serum potassium concentration at hyperglycemia. Giving insulin is the only treatment usually needed for the hyperkalemia of hyperglycemia in patients on ongoing dialysis.

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Lethal hyperkalemia has been reported with severe hyperglycemia in patients receiving ongoing dialysis.[1,2] The incidence, the association with the other laboratory abnormalities of hyperglycemia and the management of hyperkalemia in this syndrome have not been studied in detail, however. Administration of only insulin was reported to be adequate treatment for serum potassium abnormalities in cases of severe hyperglycemia of patients receiving long-term dialysis if acid-base balance does not change during treatment.[3]

We studied the serum potassium concentration in a large number of hyperglycemic episodes in dialysis patients. In this report we address the following questions: What are the frequency and clinical importance of serum potassium abnormalities? Which of the biochemical variables known to influence internal potassium balance[4] are statistically related to serum potassium concentration and to its changes during treatment of hyperglycemia of dialysis? Finally, the major issue addressed was whether or not giving only insulin is adequate treatment for elevated serum potassium concentration with or without acid-base balance changes.

Patients and Methods

We studied 15 patients with insulin-dependent diabetes mellitus (2 women, 13 men) on long-term dialysis therapy (hemodialysis 5, continuous ambulatory peritoneal dialysis [CAPD] 9, intermittent peritoneal dialysis [IPD] 1) who had hyperglycemia (serum glucose level >25 mmol per liter)* on admission or in whom hyperglycemia developed in the hospital. The 73 hyperglycemic episodes observed (1 to 11 per patient) developed 12 to 48 hours after dialysis in patients receiving hemodialysis and during dialysis in patients receiving CAPD or IPD. During treatment, dialysis was discontinued and insulin (2 to 5 units per hour) was administered either by continuous intravenous infusion or by frequent (every one to two hours) injections. All patients had oliguria, none had fluid losses through vomiting or diarrhea and none received fluid or base during treatment (the study period). Insulin administration was discontinued when the serum glucose concentration approached 10 to 15 mmol per liter. The study period lasted 8 to 25 hours.

Tonicity was computed as twice the serum sodium concentration, plus glucose concentration, in millimoles per liter.[5,6] Results are reported as range (mean ± standard deviation). The initial serum potassium concentration and change in serum potassium levels were compared with the other biochemical variables by correlation, analysis of variance and multiple linear regression, performed by Dorothy Pathak, PhD. We compared the initial with final biochemical measurements by the paired *t* test and episodes that included ketoacidosis with episodes without ketoacidosis by the nonpaired *t* test. The change in potassium value divided by the change in glucose value was compared with the initial serum potassium concentration by correlation and linear regression.[3]

*SI units (Système International d'Unités) are given throughout this article for serum glucose and potassium concentrations. The factor to convert glucose values to conventional units (mg per dl) is 0.055. To convert potassium values to the conventional milliequivalents per liter, the factor is 1.

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ABBREVIATIONS USED IN TEXT

CAPD = continuous ambulatory peritoneal dialysis
IPD = intermittent peritoneal dialysis
$Paco_2$ = partial pressure of arterial carbon dioxide

Results

Clinical presentation, frequency and degree of hyperglycemia, other biochemical measurements and outcome were similar in this study, regardless of the method of dialysis. Therefore, all hyperglycemic episodes (16 following hemodialysis, 49 during CAPD and 8 during IPD) are reported together.

Hyperglycemia usually developed in patients with severe illnesses. Seven patients had cardiac disease (acute myocardial infarction, severe congestive heart failure). Five other patients had infections—two urinary tract infections, one sepsis from leg gangrene, one sepsis from a clotted and infected bovine arteriovenous graft and one peritonitis. In four patients, hyperglycemia was related to compliance problems. In nine episodes, ketoacidosis was also present.

The most common presenting complaint was intense thirst. Dyspnea and abdominal pain temporally related to ketoacidosis disappeared with its correction. Dyspnea with radiographic findings of pulmonary edema was observed in seven other episodes and abated with correction of hyperglycemia. Neurologic manifestations, from lethargy to coma, developed during the episodes with ketoacidosis and during ten episodes in the two patients with sepsis. One of the septic patients also had a grand mal seizure (serum glucose concentration 65 mmol per liter, tonicity 321 mosm per liter). Other presenting complaints included nausea, vomiting and leg cramps. With treatment, hyperglycemic symptoms were lessened or disappeared in all patients. Seven patients, however, died days or weeks later, during the same hospital admission, of sepsis (two patients) or cardiac causes.

The serum potassium concentration at hyperglycemia (Figure 1) was below normal in 1%, in the normal range in 52% and above normal in 47% of the episodes (>6 mmol per liter in 30%). To study whether hyperkalemia was truly associated with hyperglycemia, we compared serum potassium levels at hyperglycemia with those at euglycemia (glucose 6.1 ± 0.9 mmol per liter). The potassium level at euglycemia (2 to 15 specimens per patient) did not include posttreatment values, up to 24 hours, but included all potassium values of blood specimens with glucose levels of less than 10 mmol per liter that were obtained during the same admission. The serum potassium concentration at euglycemia was (2.6 to 5.1, 4.1 ± 0.2 mmol per liter) less than the serum potassium value at hyperglycemia ($P < .001$, paired t test). Electrocardiograms were taken during several episodes and showed hyperkalemic changes frequently at serum potassium levels of greater than 6 mmol per liter, but rarely below this value. The return of the potassium towards normal levels after insulin administration resulted in disappearance of the electrocardiographic findings of hyperkalemia. In several patients, continuous electrocardiographic monitoring throughout treatment showed no life-threatening dysrhythmia.

Table 1 shows initial (at hyperglycemia) and final (posttreatment) biochemical variables, their changes during treatment and the statistical analysis. The serum potassium concentration did not change in two episodes (at 3.0 and 3.8 mmol per liter, respectively) and increased in three other episodes (3.8 to 4.1, 3.9 to 4.1 and 3.9 to 4.3 mmol per liter). A decrease in the carbon dioxide content during treatment (26 to 18 mmol per liter) was noted in the last episode. The initial potassium correlated significantly with the initial glucose, tonicity, CO_2 and arterial partial pressure of carbon dioxide ($Paco_2$). Figure 2 shows the relation between the initial potassium concentration and CO_2 content as an example of significant correlation. The mean potassium was progressively higher at progressively lower intervals of CO_2 content. Individual serum potassium values, however, varied greatly within the same interval of serum CO_2. By multiple regression, the combined initial glucose level, tonicity, CO_2 content, pH and $Paco_2$ accounted statistically for .278 (r^2) of the variability in the initial serum potassium level (Table 1). By

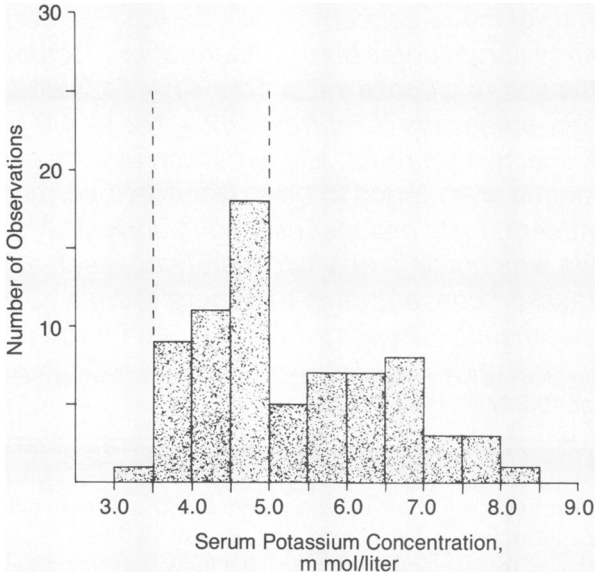

Figure 1.—The graph shows distribution of presenting serum potassium concentration in 73 hyperglycemic episodes in patients with insulin-dependent diabetes mellitus receiving ongoing dialysis. Interrupted lines indicate lower and upper normal limits.

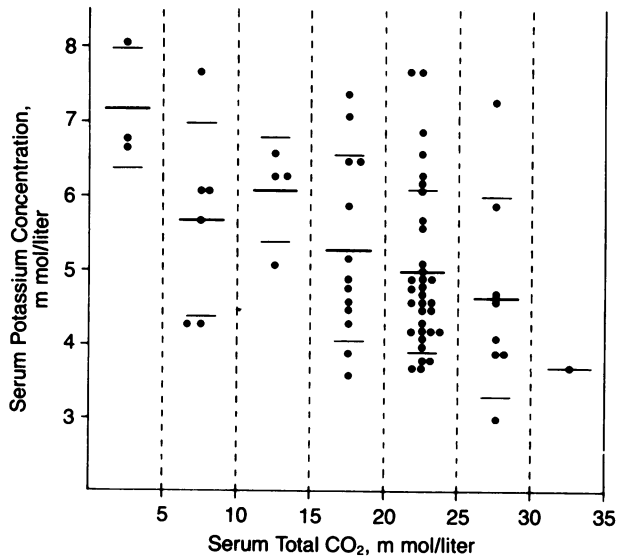

Figure 2.—The graph shows comparison of initial serum potassium concentration to initial serum carbon dioxide content in patients with hyperglycemia due to dialysis. Analysis of variance was significant ($P < .05$). Mean potassium levels (thicker horizontal lines) are higher at lower total CO_2 intervals. Within each 5 mmol per liter total CO_2 interval, however, the standard deviation (thinner horizontal lines) and the range of serum potassium levels were great.

TABLE 1.—*Initial and Final Biochemical Variables in Hyperglycemia of Dialysis*

Concentrations in Serum	Potassium, mmol/liter	Glucose, mmol/liter	Tonicity, mosm/liter	Total CO_2 mmol/liter	Arterial pH	Arterial Pco_2, torr
Initial, range	3.0 to 8.1	25 to 81	268 to 321	2 to 31	6.79 to 7.51	12.0 to 66.0
Mean±SD	5.2±1.2	41±14	293±11	20±7	7.32±0.17	30.4±11.6
r*	...	.428	.246	−.428	−.187	−.378
P†	...	.0002	.04	.0003	NS	.02
Final, range	2.4 to 5.6	2 to 25	253 to 300	16 to 33	7.32 to 7.70	14 to 40
Mean±SD	4.0±0.6	11±5	279±9	24±4	7.46±0.11	33.5±11.0
P‡	<.001	<.001	<.001	<.001	NS	NS
Change, range	−4.0 to +0.4	−12 to −70	−4 to −36	−8 to +17	−0.06 to +0.55	−26 to +26
Mean±SD	−1.2±0.4	−30±13	−14±7	+4±6	+0.14±0.13	+3.1±11.7
r*	−.850	...	...	...	...	...
r§		.663	.654	−.619	−.472	−.574
P†	.0001	.0001	.0001	.0001	.007	.0007
N	73	73	69	68	37	37

NS=no significance, Pco_2=partial pressure of carbon dioxide, SD=standard deviation

*Correlation with initial serum potassium level.
†Significance of correlation.
‡Significance of the difference between initial and final values.
§Correlation with the change in potassium concentration.

stepwise regression, only the initial serum glucose value and CO_2 content met the .150 significance level for entry into the statistical model. Combined initial serum glucose concentrations and CO_2 content accounted for .239 of the statistical variability in the initial serum potassium level.

The change in potassium level correlated significantly with the initial potassium and with the change in each of the other biochemical variables (Table 1). By multiple regression, the combined changes in all the other measurements accounted statistically for .555 of the variability in the change in potassium level. By stepwise regression, the change in serum glucose and CO_2 content met the .150 significance level and combined accounted for .488 of the statistical variability of the change in potassium. The initial potassium, change in serum glucose and change in CO_2 content accounted statistically for .891 of the variability in the change in potassium value. The effect of the initial potassium concentration on the change in serum potassium was independent of the effects of the other variables. Comparison of the change in potassium value per the change in glucose to the initial serum potassium (x axis) revealed $y = -0.060 + 0.019x$, $r = .807$ ($P < .0001$).

For the nine episodes of ketoacidosis, the initial potassium (5.9 ± 1.2 mmol per liter), glucose (56 ± 19 mmol per liter) and tonicity (301 ± 15 mosm per liter) did not statistically differ from the corresponding values of the 64 episodes without ketoacidosis; the initial CO_2 content (6 ± 3 mmol per liter), arterial blood pH (7.08 ± 0.14) and $Paco_2$ (16 ± 6 torr) were all statistically less than the corresponding values for the episodes without ketoacidosis. For the episodes with ketoacidosis, the initial potassium value did not correlate with any of the other initial variables. The final potassium (3.7 ± 0.7 mmol per liter) and change in serum potassium (-2.2 ± 0.9 mmol per liter) did not differ statistically from the corresponding values of the episodes without ketoacidosis. The change in potassium concentration correlated only with the initial potassium level ($r = .776$, $P = .014$) and marginally with the change in glucose ($r = .647$, $P = .059$). For ketoacidosis episodes, the change in the potassium concentration per the change in pH was -0.5 to -1.8 (-0.9 ± 0.5) mmol per liter per 0.1 unit.

Potassium replacement (20 and 10 mmol of potassium chloride given intravenously, respectively) was needed after the end of the study for only two patients with both ketoacidosis and protracted vomiting before presentation. The initial potassium concentration was 4.1 and 3.9 mmol per liter, respectively; the serum potassium level at the end of the study was 2.4 and 2.5 mmol per liter, respectively, for these patients.

Discussion

Patients with diabetes mellitus in whom hyperglycemia develops are at risk of hyperkalemia, even in the absence of acidosis[7-14] and even when serum aldosterone levels are normal.[15] The increased urinary excretion of potassium,[16] however, prevents severe hyperkalemia in hyperglycemic patients with intact renal function. Usually only patients with impaired renal function have severe hyperkalemia from hyperglycemia without acidosis.[8]

Diabetic patients on dialysis therapy have severely impaired renal function and are reportedly at risk of lethal hyperkalemia from hyperglycemia.[1,2] The serum potassium level, however, was in the normal range in most published reports of dialysis hyperglycemia.[17-22] In the present report, patients had normal potassium levels at euglycemia and were not receiving hyperkalemic medications. Severe hyperkalemia with electrocardiographic abnormalities, but no mortality, was found during 30% of the hyperglycemic episodes. Catabolic diseases were associated with the hyperglycemic episodes in these patients. The frequency of hyperkalemia at hyperglycemia was high, whether the patients were receiving hemodialysis or CAPD. In contrast, in routine outpatient measurements in diabetic patients, including the 15 patients of this study, we frequently found severe hyperkalemia with hyperglycemia before hemodialysis (39% of 59 episodes), but only occasionally with hyperglycemia observed in patients on CAPD therapy.[23] Kaldany and co-workers reported a 50% frequency of severe hyperkalemia in eight hyperglycemic episodes in patients receiving hemodialysis.[24]

Pathogenetically, the potential mechanisms of hyperkalemia with hyperglycemia include hypertonicity, metabolic acidosis and insulin deficiency. In hypertonicity, shifts of

intracellular potassium into the extracellular compartment[25] are independent of acid-base changes[26] and may create severe hyperkalemia at hyperglycemia.[27] Hypertonicity is usually not severe in episodes of hyperglycemia of patients receiving dialysis.[22,28] Consequently, the hyperkalemic effect of hypertonicity is usually minor in these patients. An increased blood urea nitrogen concentration creates hyperosmolality in renal failure[5] but apparently does not create hyperkalemia.[29] The distribution of urea in total body water, rather than solely in extracellular compartment, is the probable cause of the absence of hyperkalemia,[30] because there would be no fluid shifts from the intracellular into the extracellular compartment.

Metabolic acidosis also produces hyperkalemia by internal potassium shift.[31] Changes in the CO_2 content modulate the potassium level even when the extracellular pH does not change.[32] In this study, the serum CO_2 had a strong statistical correlation with the serum potassium, but hyperkalemia was not statistically different between episodes with and those without ketoacidosis. Furthermore, although the average decrease in the potassium per increase in arterial pH was consistent with published observations,[31] no statistical association between the change in the potassium concentration and that of the pH was found.

Neither hypertonicity[16] nor metabolic acidosis[3] is necessary for the development of hyperkalemia in patients with hyperglycemia. Insulin deficiency causes defective uptake of potassium by cells independently of any glucose or acid-base imbalance.[33] Insulin promotes potassium uptake by muscle cells in vitro even when the solution bathing the cells contains no glucose.[34] In patients receiving dialysis, insulin is the major mediator of cellular uptake of potassium infused intravenously.[35] We suggest that the correlations between the initial serum potassium and the other insulin-dependent initial variables, such as glucose, tonicity and CO_2 content, represent to some extent different manifestations of the same hormonal deficiency.[12,36] The combined biochemical effects, however, of lack of insulin accounted statistically for only .278 of the variability in the initial serum potassium level. Because insulin concentrations were not measured, a variation of insulin levels at presentation could account for an additional fraction of the variability in the initial potassium concentration. This fraction should be small because of the indirect evidence that initial insulin levels were low uniformly. Hyperglycemia[37] and the absence of insulin resistance in this study (administering low-dose insulin corrected the hyperglycemia in every instance) provide this indirect evidence. Therefore, factors other than insulin should be responsible for a great part of the variability in the initial serum potassium level. Potentially these factors include hormones, such as aldosterone and epinephrine,[33] uremia[4] and changes in external potassium balance. Several patients reported either protracted vomiting or consumption of large amounts of carbohydrates and potassium before admission, providing historical support for a role of external potassium balance in modulating a presenting serum potassium level.

Unlike influences on the initial potassium value, the effects of the infused insulin accounted statistically for the major part of the change in the potassium concentration during treatment. The statistical effect of the initial potassium level on the change in potassium was independent of the other variables and has clinical importance. Regardless of what factors determined the potassium concentration in the presence of hyperglycemia, insulin alone in amounts sufficient to correct the hyperglycemia restored the ability of internal potassium balance to regulate the serum potassium concentration. Thus, during treatment the decrease in the potassium value was great in patients presenting with hyperkalemia and small or none in patients presenting with normokalemia. After treatment, the potassium level was normal in most patients. Only two patients with normokalemia and ketoacidosis at presentation needed potassium replacement after treatment. Changes in serum potassium during treatment of hyperglycemia due to dialysis reported in two other studies agree with our findings. In five patients with no acid-base change during treatment, we found a significant correlation between the change in serum potassium and the initial potassium level.[3] We also calculated a significant correlation ($r = .884$, $P < .01$) between the change in potassium and the initial potassium concentration in the eight hyperglycemic episodes reported by Kaldany and colleagues.[24]

Diabetic patients receiving hemodialysis in whom hyperglycemia develops are at high risk of hyperkalemia whether or not they have associated acidosis. If there are associated catabolic states, patients on CAPD therapy in whom hyperglycemia develops are also at risk of hyperkalemia. In all hyperglycemic patients on long-term dialysis, the hypokalemic effect of the administered insulin is modulated by the initial serum potassium concentration, by the magnitude of the decrease in serum glucose and by correction of metabolic acidosis. Insulin is usually the only treatment needed for both hyperglycemia and hyperkalemia, but patients should be monitored both for signs of unstable hyperkalemia, which may dictate hypokalemic interventions faster than administration of insulin, and for the development of hypokalemia during treatment.

That insulin deficiency is a major determinant of hyperkalemia in hyperglycemia has been concluded from recent studies of different hyperglycemic states. In 142 episodes of ketoacidosis observed in 52 patients without advanced renal failure, Androgue and colleagues[38] studied the determinants of hyperkalemia by statistical methods similar to ours and concluded that insulin deficiency is the major cause of hyperkalemia. In a recent editorial,[39] the same conclusion was reached.

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